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Alison Jean Perkins
University of Richmond

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By Alison Jean Perkins

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Advisor: Dr. Penny Reynolds
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Introduction

Competitive swimmers and coaches have had varying ideas about what exercises constitute an appropriate warm-down. However, there is much agreement about what a warm-down should achieve. A good warm-down should do three things. First, a warm-down should be successful in returning the swimmer's elevated heart rate to the resting heart rate level in a relatively short period of time. Second, the warm-down should flush lactic acid out of the muscles as quickly as possible, and restore blood lactate to baseline levels. Finally the warm-down should contribute to improved athletic performances.

Heart rate is an indication of how hard the heart is working. During exercise, muscle cells require more oxygen in order to produce sufficient ATP. When ATP is hydrolyzed into phosphate and ADP, energy is released. This energy is necessary for muscle contraction as well as many other physiological processes that occur during exercise. Since ATP is needed during these times of exertion, the mitochondria within the muscle cells must work hard to constantly replenish this depleting supply of energy. When oxygen is present, pyruvate (the product of glycolysis) can enter the Kreb's Cycle and continue on through the electron transport chain. Oxygen acts as the final electron acceptor in the electron transport chain, which simultaneously produces large amounts of ATP (1).

During aerobic respiration, blood is oxygenated in the lungs. Oxygenated blood from the lungs enters the left atrium, and then fills up the left ventricle. The highly muscular walls of the left ventricle allow for a strong enough contraction to force blood through the vascular system into the extremities. Oxygen, which is carried through the body attached to hemoglobin in red blood cells, is released into tissues that contain a lower partial pressure of oxygen. In this scenario, the tissue is muscle tissue. At this point, oxygen dissociates from hemoglobin and associates with myoglobin. Myoglobin then transports oxygen to the appropriate cells.

When the muscle cells use up ATP faster than it is replenished naturally, the heart is triggered to work harder. In order to increase the amount of oxygen the body receives, the heart needs to increase the rate of blood flow. Consequently the time between heart beats decreases, which means that the heart is beating faster. In other words, the number of heart beats per minute increases. This explains why an athlete's heart rate increases with exercise. Heart rate is controlled by the modulating influence of the autonomic system on the spontaneous firing of the pacemaker cell within the sinoatrial node. The sympathetic nervous system increases spontaneous firing, while the parasympathetic system depresses firing of the pacemaker cells (2). The interval between cardiac action potentials determines the heart rate. This interval depends on the rate of depolarization of the pacemaker potential. The

faster depolarization brings a membrane to a firing level, the greater the frequency of firing that occurs on the cardiac pacemaker cell membranes.

At a certain level of exercise, the heart is not capable of beating any faster. As a result, within muscle cells, there is insufficient oxygen to allow pyruvate to enter the Kreb's Cycle and yield large amounts of ATP. Therefore, the body substitutes aerobic respiration (utilizing oxygen) for anaerobic respiration. Under normal (non-exercising conditions) the body produces lactic acid constantly, and it is shuttled from one part of the body to another to serve as a source of fuel. For example, the heart's energy source during exercise is lactic acid (3). In anaerobic respiration pyruvate and NAD+ are converted into lactic acid and NADH. The NADH is recycled into glycolysis so that at least a little ATP can continue to be made (a net of 2 ATPs are produced from glycolysis). The reason lactic acid accumulates in the muscles and causes the achy feeling many athletes experience with a strenuous work-out is because, it is no longer efficiently removed from the muscles by the blood stream. Once the body returns to normal levels of exertion, the heart begins to beat normally and lactic acid is more efficiently removed from the muscles and converted back into pyruvate. With oxygen now available, pyruvate can enter the Kreb's Cycle followed by the electron transport chain to yield larger quantities of ATP.

It is desirable to remove lactic acid from the muscles as efficiently as possible following exercise. The removal of blood lactic acid is regulated by several factors. These factors include: the efflux rate of lactate from muscles to blood (4); the blood flow rate (5); muscle capillary density (6); and the metabolic removal by the heart, liver, and muscle (7). The exercising muscle is responsible for 55-70% of total lactate removal due to oxidative processes (7). During exercise recovery (a warm-down) the optimal efflux rate of lactate from muscles to the blood is maintained. Blood lactate levels are diluted due to the increased blood past the exercising muscular cell membrane. This is countered by the muscle lactate transporter that moves lactic acid from an area of high concentration (muscle) to an area of lower concentration (blood) where it is transported for removal to the heart, liver, or muscle (8)(9).

In this study, we aimed to determine which swimming warm-down would remove muscular lactic acid more efficiently (a gradually decreasing intensity warm-down or a constant intensity warm-down). It was hypothesized that the gradually decreasing intensity warm-down would more efficiently remove lactic acid, because the body would be more able to adjust to a gradually decreasing blood output than to an abrupt decrease.

In addition to determining the effect of two different warm-downs on the removal of lactic acid from the muscles, this study was intended to determine the effect of such warm-downs on heart rate recovery. The

period of resuming normal heart activity following exercise is called heart rate recovery. One study showed that after periods of heavy exertion, the heart rate recovery follows a distinct pattern containing two phases (9). An exponential drop in heart rate (fast phase) characterizes the first phase, and the second period is characterized by a slower decline in heart rate to the resting heart rate (slow phase) (10). In comparison to untrained swimmers, trained swimmers exhibit an earlier deviation from the fast phase, and reach the second phase base line sooner than an untrained swimmer (10). In other words, the first phase slope of recovery is greater in trained swimmers than untrained swimmers. Consequently it can be assumed that a quicker heart rate recovery after a work out is indicative of fit swimmers. As it can be seen, it is desirable to return the heart to its resting heart as efficiently as possible following exercise.

In this experiment we determined the correlation between two different warm-down protocols and the ability of the heart to efficiently return to resting heart rate conditions. It was hypothesized that a decreasing intensity warm-down would result in more efficient heart rate recovery. Such a warm-down would be expected to put less stress on the heart, allowing a more efficient return of the heart to normal levels of exertion.

Materials and Methods

Twelve female swimmers, between the ages of 18 and 22, from the University of Richmond Varsity Swimming and Diving Team were the subjects of this study. All of these swimmers received a notice explaining their rights and the details of the study. All swimmers were asked to sign a consent form allowing permission to perform the stated tests. Although the risks are minimal, each swimmer was warned of all the possible risks involved in participating in this study. Subjects were warned that there was a slight chance that they would feel a twinge of pain from the finger prick. The only other risk to the swimming subjects included the normal risks of swimming (including drowning, cardiac arrest, etc.). Since the subjects were experienced swimmers, I anticipated these risks were quite low. Per chance that the athletes encountered medical difficulty, a certified first aid/CPR person was always available on the pool deck during the experimentation. In addition a first aid kit is always available in the pool office as well as an emergency telephone on deck. Finally, a trainer was available in the training room, not far away from the pool. To ensure sterility in the testing, the skin was washed thoroughly with an alcohol pad to remove bacteria, sweat, and chlorine. In addition, the examiner always wore fresh latex gloves and used new lancets for every finger prick to prevent the transmission of possible diseases. These gloves, as well as the alcohol swabbing pads, were disposed of in a red hazardous waste plastic bag.

The sharp needles were disposed of in a red, plastic, sharp, hazardous waste bin. The hazardous waste bin and bag were accepted by the biology department at the University of Richmond and taken for proper disposal. This disposal procedure abides by OSHA standards for the disposal of materials used in blood tests.

To begin the experiment all the swimmers were asked to determine their resting level heart rate and lactic acid level. To determine their resting heart rate, the swimmers were asked to record their heart rate first thing in the morning for one week. They measured the amount of beats in thirty seconds and then multiplied by 2 to obtain their resting heart rates per minute. Resting lactic acid levels were recording using the Accusport Portable Lactate Tester when they arrived on the pool deck, prior to any aquatic exercise. The swimmers' maximal heart rates and lactic acid levels were established by requiring that each swimmer perform an exhaustive swim designed after the tethered swim in the study performed by (9). Swimmers swam with stretch cords attached to their waists until the swimmers reach exhaustion. Exhaustion was defined as the point when the swimmer could no longer maintain a stationary position in the water and was pulled-back by the resistance of the stretch cords (9). It was determined through previous experimentation that maximal blood lactate levels occur between 2 and 4 minutes of quiet rest following maximal exercise (9). Consequently, at this time of rest following the exhaustive swim, the swimmer's maximal

blood lactate level was determined using the Accusport Portable Lactate Tester. In addition, maximal heart rates were recorded at this time using Minimitter Dataloggers.

For each swimmer, 85%, 75%, 65%, and 50% of their maximal heart rate was calculated using the following formula:

%(Max H.R - Min H.R.) + Min H.R. = Percentage H.R.

These calculations of the percentage heart rates were used to guide the swimmers to swim at certain intensities in their warm-down. The University of Richmond Swimming and Diving Coach claimed that the swimmers know how to control their swimming pace to reach a specified target heart rate.

Two different warm-down protocols were tested. In each protocol, the swimmers swam 4 one-hundred yard distances with a 30 second rest between sets. In the first protocol (Protocol A), each set was swam at 50% maximal heart rate. This warm-down was designed to keep the swimming intensity at a constant exertion throughout the warm-down. In the second protocol (Protocol B), the sets were swum at 85%, 75%, 65%, and 50% maximal heart rate respectively. This warm-down was designed to create a swimming pace that was gradually decreasing in intensity. Each day of testing, four random women were selected to participate in the study for that particular day. On these days, two

women swam the Protocol A warm-down, while the other two swimmers swam the Protocol B warm-down. All four swimmers staggered their warm-downs (2 minutes between each swimmer), so that lactate test could be performed immediately following the warm-down. So that the swimmers did not sit around on the pool deck waiting for their turn to begin their designated warm-down regimen, they waiting swimmers were given addition work-out sets to keep their heart rates up at practice level. Between each 100 m swim, during the 30 seconds rests, swimmers verified that they were swimming at the right speed to control maximal heart rate percentages by reading their Minimitter Dataloger watches and catching their pulse. Adjustments were made in the swimmers' paces according to any deviation from the target percentage heart rate. Following the protocol warm-down, blood lactate levels were immediately checked using the Accusport Portable Lactate Tester. In addition, the initial heart rate was also recorded. Swimmers were asked to sit quietly on the pool deck until heart rate and lactate levels return to resting levels (or leveled off). Heart rate will be recorded every 60 seconds and lactic acid every 5 minutes. In order to protect the interest of the participating athletes, the lactate finger pricks were limited. A five minute (or ten minute) finger prick was only administered when lactate levels were high after the time zero prick. If the lactate levels were low (as compared to their respective resting lactate levels) the swimmers were spared a second finger prick. Throughout the three weeks of testing, most

swimmers performed Protocol A and B warm-downs twice. Because of the time constraints, some swimmers did not complete other warm-downs twice.

Following the collection of the data, statistical analysis was performed. Heart rate data for each warm-down protocol was analyzed separately. A repeated-measures analysis of variance with a polynomial test for trend was used (11). To estimate the change in heart rate over the warm-down period, a fitted least squares regression on ln-transfer data was performed (9).

Preliminary descriptive analysis (means, standard deviation) of lactate data indicated that variability between individual swimmers was too large to permit the use of inferential statistics. Accordingly, data was examined on a case-by-case basis using information on maximum and resting lactate levels, in addition to lactate concentrations measured during recovery. Finally, to estimate the change in lactate over the warm-down period, a fitted least squares regression on ln-transfer data was performed (9).

Results

Protocol A (constant intensity warm-down) resulted in a decrease in heart rate, which consisted of an exponential "fast phase" and a linear "slow phase"; similar to what was observed in the study by Richardson et al (Figure 2) (9). Following this warm-down, the average heart rate was around 130 bpm (Figure 1). Figure 2 shows that heart rate decreased rapidly initially (slope = 0.113) in the fast phase. Then heart rate gradually leveled off to about 85 bpm (Figure 1). This slow phase of recovery following Protocol A had a slope of 0.0125. In a Test of Within-Subjects Contrasts, for Protocol A, heart rates entered the linear phase after approximately 4 minutes ($p>0.05$) (Table 4).

In contrast, Protocol B (decreasing intensity warm down) resulted in a step-like decrease in heart rate that consisted of 2 exponential decreases in heart rate separated by two linear plateau phases (Figure 2). Like Protocol A, the average heart rate was around 130 following Protocol B (Figure 1). Figure 2 showed that heart rate decreased rapidly at first (slope = 0.867) in the first fast phase. Soon after heart rate temporarily leveled off in the first slow phase (slope = 0.015). The second fast phase following Protocol B did not appear to be as fast as the first (slope = 0.04). In the final slow phase, heart rate leveled-off (slope= 0.01) at about 92 bpm (Figure 1 and 2). In a Test of Within-Subjects Contrasts for Protocol B, any visible change in heart rates were insignificant ($p> 0.05$) after five minutes (Table 4).

A t-test (comparing slopes over the entire range of heart rate recovery) showed that the two warm-downs do not restore heart rate differently. Protocol A had an overall slope of 0.0489, and Protocol B had an overall slope of 0.0384. The calculated t value (0.687) was less than the t critical value (2.145)(11). Therefore, the null hypothesis (no difference in slopes) was accepted.

It is also relevant to note that a Test of Between-Subjects Effect was performed to see if there was significant difference in heart rate recovery among the tested athletes. The results showed that there was little significance in heart rate recovery following Protocol A ($p=0.00$) and Protocol B ($p=0.00$) between the athletes.

Although there was a difference between heart rate recovery following Protocol A and B, there was not much difference in efficiency of muscular lactic acid removal after performing these two warm-downs. Table 5 showed that the mean lactic acid level following Protocol A at t=0 was 2.1583. This was lower than the mean lactic acid level following Protocol B at t=0 (2.4333). Although these numbers differed, the standard deviations were so tremendous (0.6895 and 0.8637 respectively) that this difference could hardly be recognized as significant ($p>0.05$). Graphically, there did not appear to be significant difference in the rate of lactic acid removal in comparing Protocol A and Protocol B (Figure 4). The slope for Protocol A (0.0273) was not significantly different from the slope for Protocol B (slope = 0.0303). Figure 3 showed

that, although the rate of lactic acid removal was equal following the warm-downs, the rate of lactic acid removal was greater for Protocol A during the actual warm-down. This was evident, because lactate levels were overall lower following this warm-down (Figure 3).

Discussion

Overall, it appeared that Protocol A (a constant intensity warm-down) was more efficient at restoring resting heart rates and resting lactic acid levels. Protocol A followed the expected two-phase pattern of heart rate recovery for physically fit swimmers (Figure 1)(9). The question then was, why did Protocol B result in a step-like pattern of heart rate recovery (Figure 3)? Perhaps, the decreasing intensity warm-down maintained the athletes' intensity at too high a level for an effective warm-down. This is because the swims in this warm-down were at 85%, 75%, 65%, then 50% of maximal heart rate as compared to the lower 50% maintained in Protocol A. A step-like heart rate recovery pattern indicates a disruption to normal heart rate recovery. After Protocol B, the heart gradually decreased its output, remained constant, it decreased a little bit more, then remained constant again. It is almost as if the sudden cease of warm-down activity was a shock to the system as if there were no warm-down at all. Further studies to see if the heart rate recovered similarly when no warm-down is used would be interesting. Physiologically, it appears that a constant intensity warm down has the beneficial combination of keeping heart rate high enough to not alarm the heart during the warm down itself, but low enough to not alarm the heart following the warm down in the recovery period.

Another possibility is that the decreasing intensity warm-down was reflected in the heart rate recovery (step-like pattern) just as the constant

intensity warm-down was reflected in the heart rate recovery (exponential decrease). The heart worked hard at the greater percentage heart rates (85%, 75%, and 65%). Then during the 30 second breaks between 100 meters sets the heart slowed down rapidly. These pauses may be reflected in the heart rate recoveries "fast phases" where the heart slowed down quickly. The following "slow phase" could be a reflection of the heart working hard during the 100 meters swims. Figure 4 shows that the slope of this second fast phase was less than the first, which could be explained by the fact that the heart recovered more slowly when the heart rate was down than when it is up. This pattern could be seen in the Protocol A heart rate recovery curve, where recovery was initially rapid and gradually slowed down as heart rate decreased (Figure 1).

A comparison of initial (fast phase) recoveries following each warm-down showed that Protocol A was initially much more efficient at decreasing heart rate (Figure 2). This was evident by comparing these initial slopes. The t-test, comparing the overall slopes of each Protocol, showed that there was no difference in the overall rate of recovery following these warm-downs. The most significant difference was seen in the baseline heart rate level following each warm-down. Protocol A returned the heart to a lower level than Protocol B (Figure 1). For this reason, a constant intensity warm-down appeared to be better at returning the heart to resting levels.

It is important to consider the variability among the athletes resting, maximal, and recovering heart rates. It is commonly said that the more physically fit a person is, the lower their resting heart rate should be. It was surprising how high several of the swimmers' heart rates were. For example, the resting heart rate of swimmer C was 84 bpm, compared to swimmer E whose heart rate was 60 bpm (Table 1). This variability could be attributed to the fact that many people's heart rates increase when they are counting it because of anticipation. Several of the swimmers actually mentioned that their heart rates were quite low (observing the Minimitter Datalogger watch), but when it came time to record the heart rate value, it went right up because of this anticipation. In addition, variability among maximal heart rates could be attributed to variability in perceived effort of the swimmer.

The rate of removal of lactic acid from the muscles appeared to be unrelated to the swimmers' warm-downs, but it does seam to be related to the amount of lactic acid in the muscles initially after the warm-down. A constant, low intensity, warm-down is more effective at removing lactic acid from the muscles during the actual warm-down than a decreasing intensity warm-down. The gradually decreasing intensity warm-down maintained a high heart rate much longer than the constant intensity warm-down. As a result, ceasing physical activity following the decreasing intensity warm-down was similar to ceasing activity following little or no warm-down. If a swimmer were to swim a fast sprint, and

then cease further movement, the swimmer would experience severe cramping due to the muscular accumulation of lactic acid. The anaerobic system (in which pyruvate is converted into lactic acid) would still be working at high levels. In contrast, by warming-down, an athlete gradually returns to aerobic respiration and can prevent the painful (and abrupt) accumulation of lactic acid in the muscles. Instead the body flushes lactic acid out of the muscles, because anaerobic respiration is no longer necessary. Although lactic acid may be removed from the muscles at a constant rate, an effective warm-down returns the body to aerobic respiration sooner, allowing lactic acid levels to decrease sooner.

As can been seen in Table 1, there was tremendous variability among the athletes' maximum and resting lactate levels. There were several explanations for this observation. Aside from the physical symptoms of achy muscles, a swimmer experienced mental symptoms that limit their ability to push themselves further physically. Each swimmer had a different perception of what was exhaustion; some swimmers mentally could handle more exertion than others can. For example, after the exhaustive swim, swimmer B had a very high lactate level (6.7 mM), but swimmer I had a very low lactate level (2.4 mM) (Table 1). Perhaps swimmer I mentally could not handle any more physical exertion, or she may have lacked the motivation. Psychological visualization and training could enhance the swimmer's ability to work harder. The other possibility is that swimmer I had a much greater

aerobic capacity than swimmer B, and had the ability to transport more oxygen to muscle cells during short spurts of physical activity. Interestingly, swimmer B was a distance swimmer (who trained for endurance), whereas swimmer I was a sprinter who trained for short, rapid swims. It might be beneficial for sprinters to increase the amount of endurance training, because this way they may be able to push themselves further at a greater intensity without feeling the achy accumulation of lactic acid in the muscles.

The observable differences seen among the swimmers resting lactate levels could be simply biological differences, or they could be due to variation due to testing humans. It was difficult to regulate the activities of the swimmers prior to their entering the pool deck. Some swimmers may run to the pool, because they are afraid of being late for practice. For example, swimmer J had an exceptionally high initial lactic acid level (5.6 mM). This could also be attributed to the fact that she had just recently recovered from asthmatic bronchitis and was presumably taking medication for this problem. Others may have low lactate levels because they had been sitting in the pool office studying prior to practice. For example swimmer E had a lactate level of 1 mM for her resting level (Table 1).

In addition to the resting and maximum lactate variation, there was quite a bit of variation among the swimmers with their time= 0 lactate levels following the warm-downs. For example, following Protocol

A swimmer A had lactate levels between 3 mM - 3.3 mM. In contrast, swimmer G had lactate levels between 1.6 mM - 1.8 mM (Table 4). Although the mean was 2.21583 mM, the standard deviation was great (0.6895 mM) (Table 5). Similar variability was observed among the swimmers with their t= 0 lactate levels following Protocol B. Again, this variability could be the result of different efforts put into the work-outs or warm-downs. Each swimmer had different levels of motivation and psychological endurance. Also, interestingly many swimmers got their lactate levels lower than the resting level following the warm-downs. For example, swimmer K had a resting lactate of 2.2 mM, but her lactate immediately following Protocol A was 1.7 mM. The body maintains a certain level of lactate in the blood despite exercise. Perhaps a warm-down removed more lactate than at resting level because the removal system was working hard, then it gradually returned to this resting level. It was also possible that lactate levels were too high for the resting time, because the athletes were moving around too much prior to practice. The first explanation seams more likely than the second one, because a warm-down is physical movement as well.

Because of the extremely small sample size, the difference observed was not completely reliable. It would be beneficial to re-run this experiment with more swimmers (and to compare any difference between distance swimmers and sprinters). It would be hypothesized that distance swimmers recover better from a decreasing intensity warm-

down than a constant exertion warm-down. This is because distance swimmers do not go into anaerobic respiration as much as sprinters do.

In this experiment, it was expected that a decreasing intensity warm-down would more gradually return the body to aerobic respiration. Although it appeared to be true (as seen in heart rate recoveries) that a decreasing intensity warm-down more gradually returned the body to resting conditions, it was not the most efficient method of recovery. The most efficient recovery was that which occurred fastest. A constant intensity warm-down appears to recover resting heart rate and lactate levels more quickly. Something to consider, though, is that perhaps the most efficient recovery is not the best for the body.

Appendix

Table 1. Resting/Maximum Heart Rates

subject	type	rest H.R (beats/min)	max.H.R. (beats/min)	50% H.R. (beats/min)	65% H.R. (beats/min)	75% H.R. (beats/min)	85% H.R. (beats/min)	rest lact. (mM)	max. lact. (mM)
A	distance	84	160	122	133	141	149	1.9	6.3
B	distance	68	191	130	148	160	173	2.1	6.7
C	sprinter	84	187	136	151	161	172	2.4	5.8
D	sprinter	62	178	120	137	149	161	2.9	5.3
E	sprinter	60	170	115	132	143	154	1	2.3
F	sprinter	70	169	120	134	144	154	2.8	5.4
G	sprinter	65	171	118	134	145	151	1.4	6.4
H	sprinter	83	177	130	144	154	163	1.3	5.7
I	sprinter	63	146	105	117	125	134	2.5	1.3
J	sprinter	67	175	121	137	148	159	5.6	2.4
K	sprinter	69	182	125	143	154	165	2.2	7
L	sprinter	66	160	113	122	137	146	1.8	4.3

Table 2. Heart rate recovery following Protocol A

Constant Intensity Warm-down

subject	type	trial	t=0 min	t=1 min	t=2 min	t=3 min	t= 4 min	t=5 min	t=6min	t=7 min	t=8 min
A	distance	a	128.00	116.00	106.00	100.00	95.00	92.00	90.00	88.00	86.00
		b	124.00	107.00	98.00	88.00	85.00	89.00	86.00	80.00	83.00
B	distance	a	147.00	130.00	132.00	126.00	122.00	120.00	121.00	121.00	124.00
C	sprinter	a	130.00	109.00	107.00	97.00	92.00	85.00			
		b	142.00	115.00	128.00	108.00	105.00	95.00	106.00	92.00	87.00
D	sprinter	a	142.00	100.00	94.00	82.00	76.00	68.00			
		b	124.00	103.00	98.00	101.00	95.00	92.00	98.00	86.00	76.00
E	sprinter	a	116.00	116.00	70.00	70.00	70.00	58.00			
F	sprinter	a	128.00	109.00	100.00	100.00	91.00	92.00	95.00	95.00	94.00
		b	122.00	106.00	102.00	99.00	97.00	93.00			
G	sprinter	a	134.00	122.00	121.00	110.00	101.00	95.00	91.00	82.00	90.00
		b	116.00	106.00	95.00	98.00	91.00	85.00	78.00	83.00	75.00
H	sprinter	a	132.00	102.00	102.00	100.00	91.00	104.00	102.00	108.00	93.00
I	sprinter	a	114.00	90.00	86.00	81.00	76.00	73.00	75.00	74.00	75.00
J	sprinter	a	136.00	110.00	96.00	77.00	75.00	78.00	70.00	75.00	76.00
K	sprinter	a	130.00	118.00	93.00	78.00	89.00	78.00	73.00	78.00	
L	sprinter	a	123.00	101.00	100.00	93.00	88.00	88.00	86.00	83.00	85.00
		b	115.00	98.00	87.00	81.00	80.00	82.00	83.00		

Table 3. Heart rate recovery following Protocol B
Decreasing Intensity Warm-down

subject	type	trial	t=0 min	t=1 min	t=2 min	t=3 min	t= 4 min	t=5 min	t=6min	t=7 min	t=8 min
A	distance	a	131.00	108.00	106.00	96.00	96.00	98.00	87.00	102.00	90.00
B	distance	a	142.00	113.00	98.00	98.00	88.00	79.00	80.00	93.00	92.00
		b	132.00	93.00	92.00	91.00	88.00	92.00	87.00	90.00	90.00
C	sprinter	a	135.00	110.00	109.00	108.00	103.00	106.00	97.00	96.00	95.00
		b	140.00	124.00	123.00	114.00	104.00	104.00	101.00	100.00	100.00
D	sprinter	a	127.00	117.00	115.00	114.00	112.00	112.00	110.00	108.00	108.00
		b	126.00	105.00	81.00	80.00	83.00	82.00	80.00	76.00	76.00
E	sprinter	a	126.00	89.00	97.00	88.00	90.00	85.00	83.00	72.00	
		b	114.00	68.00	76.00	64.00	60.00				
F	sprinter	a	135.00	113.00	118.00	120.00	115.00	95.00	102.00	103.00	101.00
		b	125.00	106.00	99.00	97.00	91.00	91.00	91.00	90.00	
G	sprinter	a	136.00	113.00	100.00	104.00	110.00	92.00	89.00	89.00	88.00
		b	136.00	133.00	105.00	114.00	107.00	99.00	95.00	91.00	95.00
H	sprinter	a	136.00	119.00	114.00	107.00	111.00	104.00	100.00	95.00	84.00
I	sprinter	a	120.00	107.00	85.00	88.00	90.00	73.00	77.00	81.00	74.00
J	sprinter	a	132.00	117.00	83.00	91.00	88.00	86.00	90.00	85.00	90.00
		b	124.00	114.00	108.00	114.00	108.00	100.00	98.00	103.00	94.00
K	sprinter	a	130.00	85.00	89.00	78.00	83.00	87.00	80.00	87.00	87.00
L	sprinter	a	118.00	95.00	93.00	88.00	80.00	89.00	89.00	92.00	84.00

Table 3. Lactic acid removal following the designated warm-downs

			constant intensity warm-down (Protocol A)			decreasing intensity warm-down (Protocol B)		
subject	type	trial	t=0 min	t= 5 min	t=10 min	t=0 min	t=5 min	t=10 min
A	distance	a	3.3	2.6		4.6	3	
		b	3	1.9		3.3	2.2	
B	distance	a	1.8			1.1		
		b				1.4		
C	sprinter	a	1.6			2.1	1.6	
		b	2.2			1.8		
D	sprinter	a	1.5				4.7	3.3
		b	2.9	2.4		1.9		
E	sprinter	a	1.5			2.5		
		b				1.2		
F	sprinter	a		1		2.4		
		b	1.8			1.2		
G	sprinter	a	1.6			0.9		
		b	1.8			2.5		2.2
H	sprinter	a	1.8			1.5		
		b	1.4					
I	sprinter	a	1.2			1.6		
J	sprinter	a	1.9			1.7		
		b				4.3	3	
K	sprinter	a	1.7			3		
		b				3.3	2.4	
L	sprinter	a	2.9	2.1		2.5		
		b	2.2					

Table 4. Significant differences in heart rate folowing Protocol A and Protocol B.

time (min.)	Protocol A p value	Protocol B p value
0	0	0
1	0	0
2	0	0
3	0.006	0
4	0.109 *	0.002
5	0.473 *	0.015
6	0.816 *	0.441 *
7	0.632 *	0.889 *
8	0.657 *	0.442 *

shows significant differnce between heart rate at this time as compared to the previous minute ($p > 0.05$).

Table 5. Mean lacate and standard deviation following Protocol A and Protocol B.

Warm-down	Mean (mM)	Standard Deviation	N
Protocol A	2.1583	0.6895	12
Protocol A	1.8833	0.3904	12
Protocol B	2.4333	0.8637	12
Protocol B	2.0917	0.5071	12

Table 6. Lactic acid removal following the designated warm-downs

			constant intensity warm-down (Protocol A)			decreasing intensity warm-down (Protocol B)		
subject	type	trial	t=0 min	t= 5 min	t=10 min	t=0 min	t=5 min	t=10 min
A	distance	a	3.3	2.6		4.6	3	
		b	3	1.9		3.3	2.2	
B	distance	a	1.8			1.1		
		b				1.4		
C	sprinter	a	1.6			2.1	1.6	
		b	2.2			1.8		
D	sprinter	a	1.5				4.7	3.3
		b	2.9	2.4		1.9		
E	sprinter	a	1.5			2.5		
		b				1.2		
F	sprinter	a		1		2.4		
		b	1.8			1.2		
G	sprinter	a	1.6			0.9		
		b	1.8			2.5		2.2
H	sprinter	a	1.8			1.5		
		b	1.4					
I	sprinter	a	1.2			1.6		
J	sprinter	a	1.9			1.7		
		b				4.3	3	
K	sprinter	a	1.7			3		
		b				3.3	2.4	
L	sprinter	a	2.9	2.1		2.5		
		b	2.2					

Figure 1. Heart rate recovery pattern following Protocol A and Protocol B

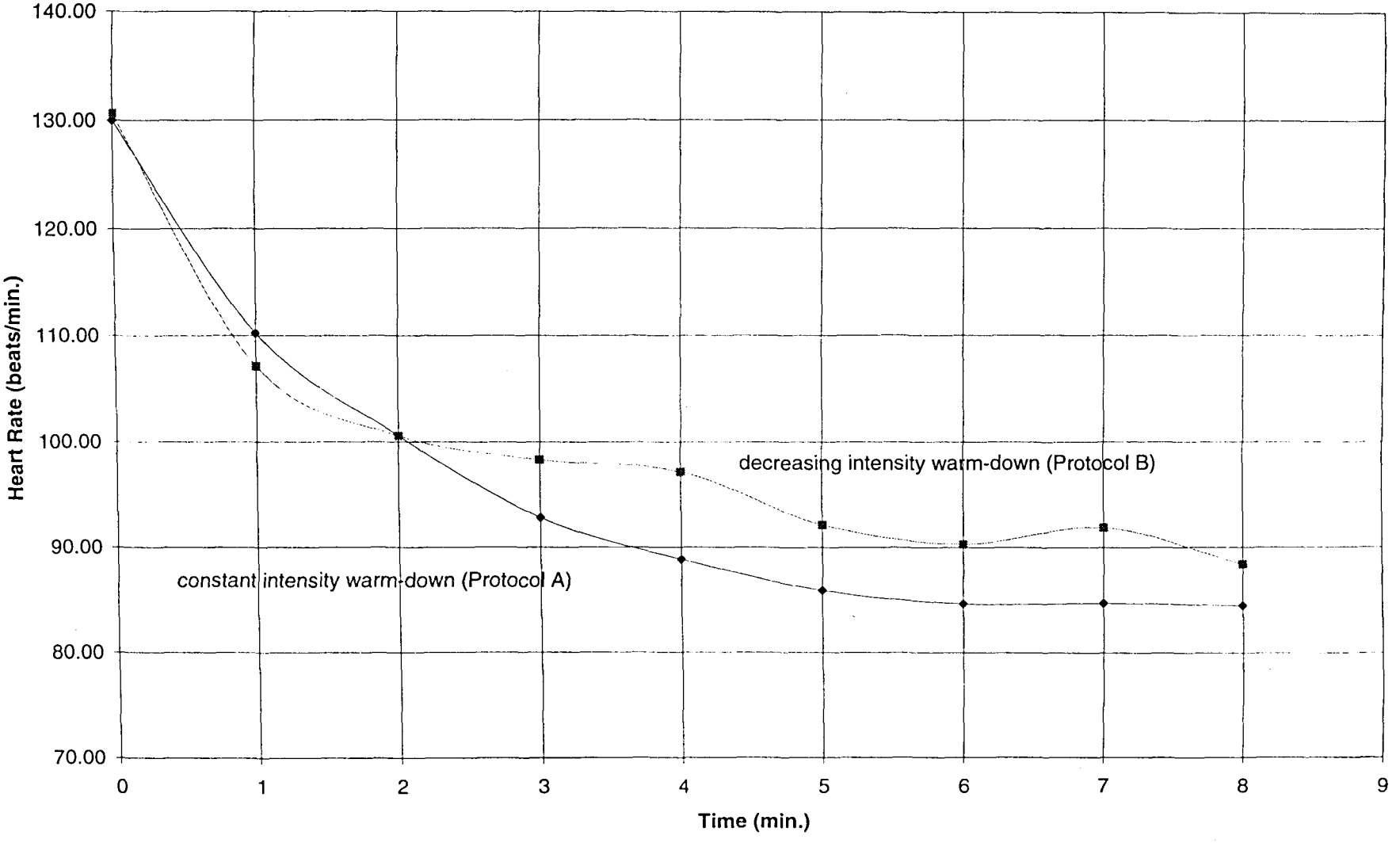


Figure 2. Fast and slow phase heart rate recovery following Protocol A and Protocol B

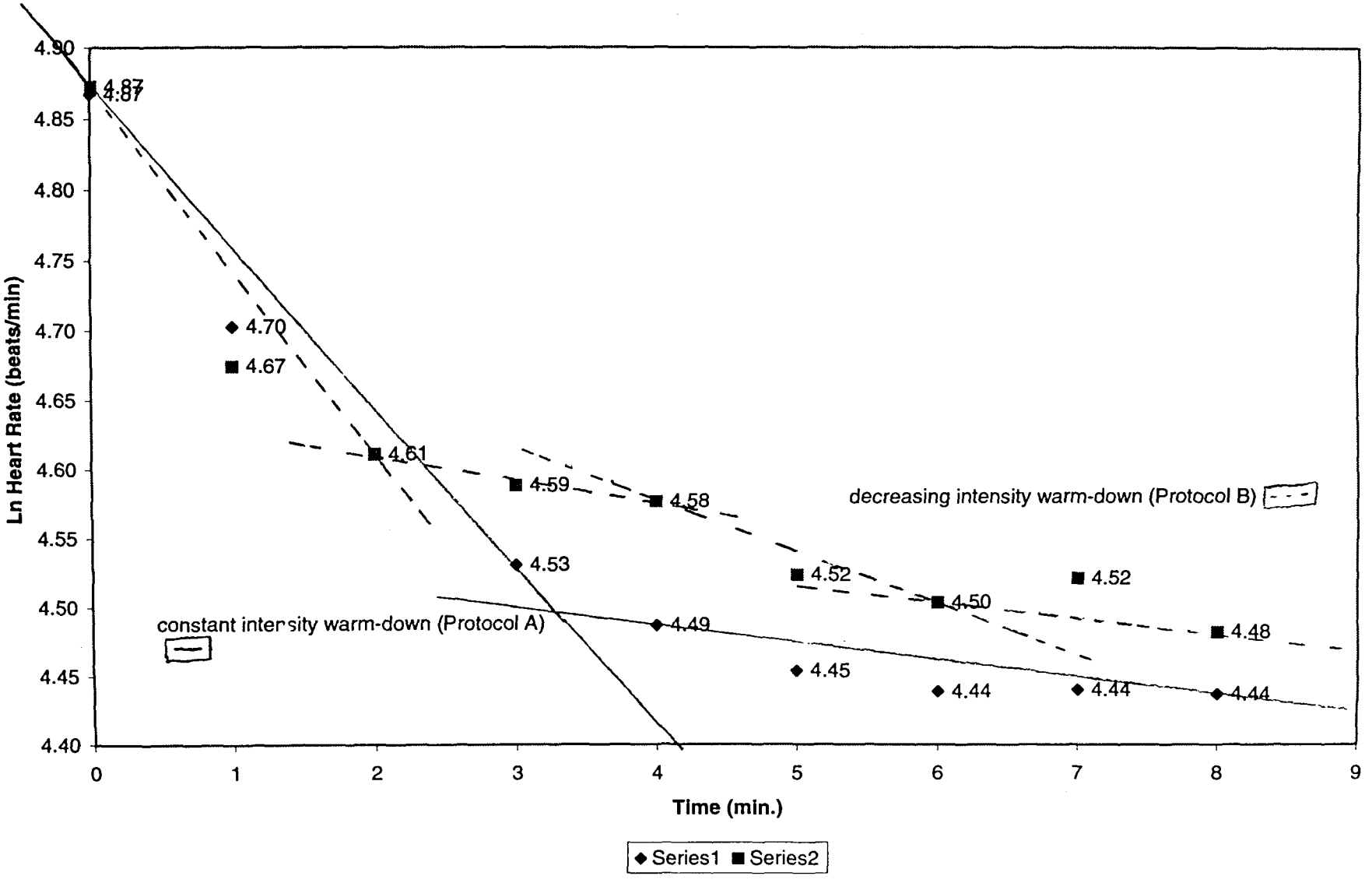


Figure 3. Muscular lactate clearance following Protocol A nd Protocol B

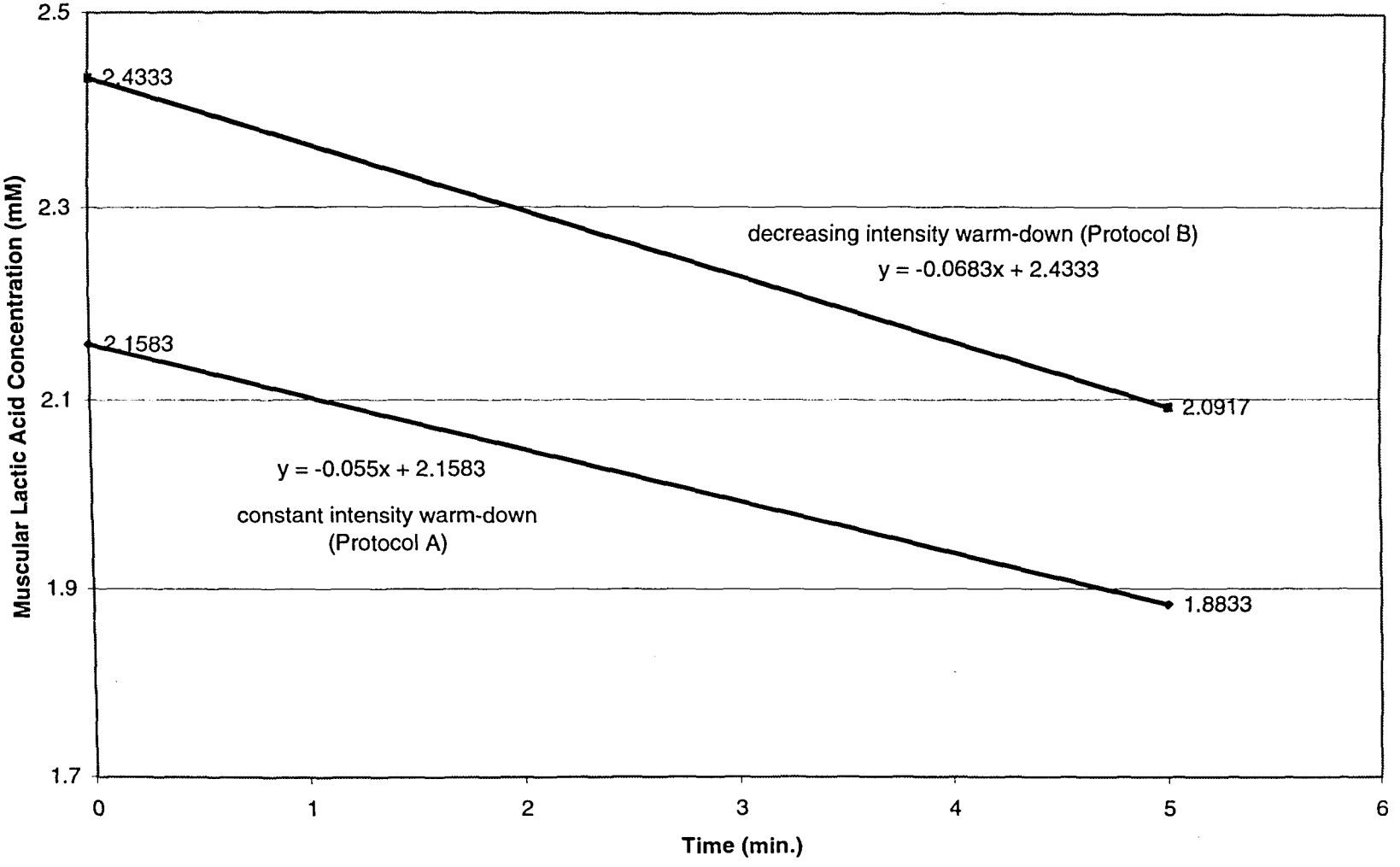
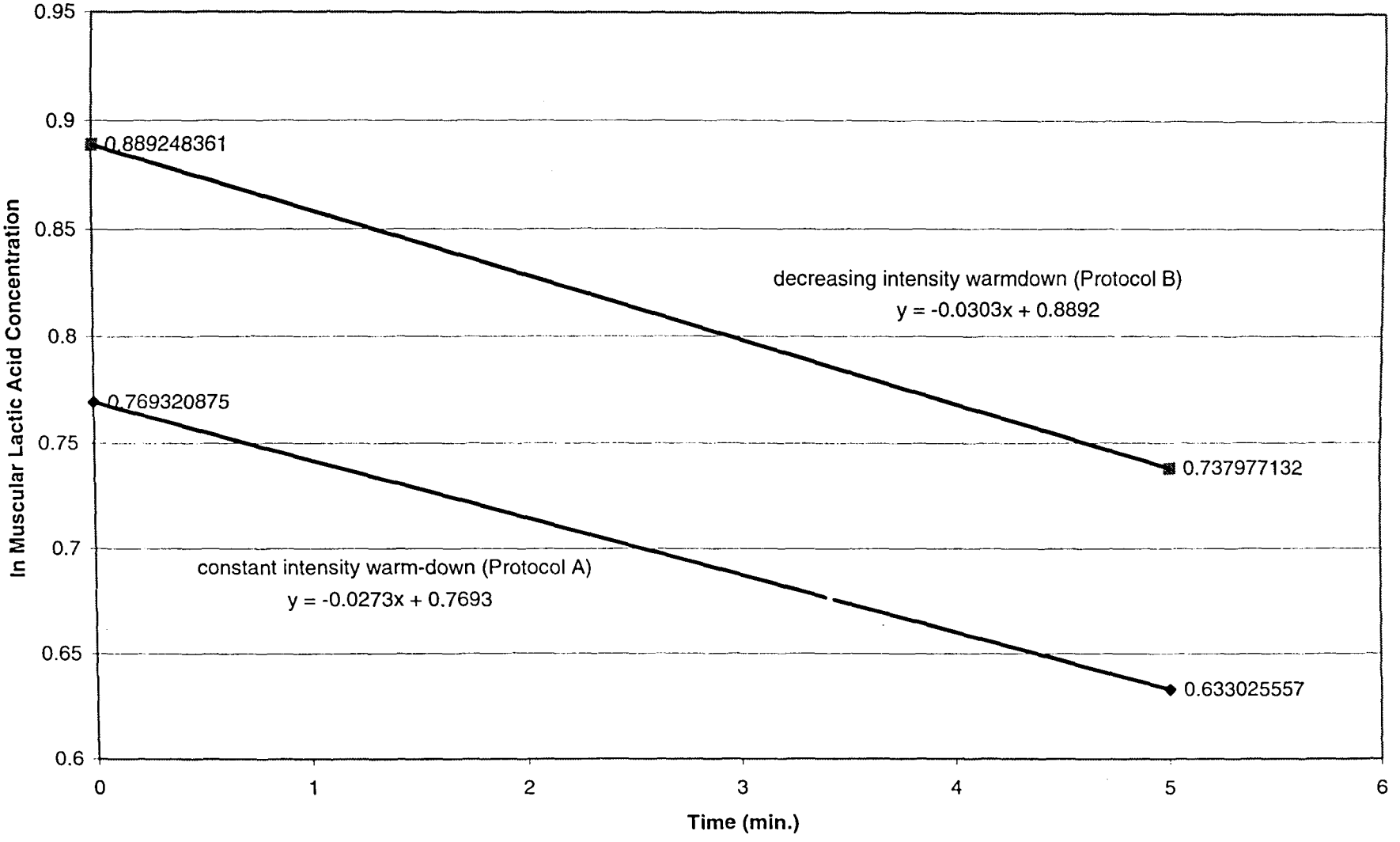


Figure 4. Rate of lactic acid clearance following Protocol A and Protocol B



Literature Cited

1. Kleinsmith, Lewis J., and Valerie M. Kish. Principles of Cell and Molecular Biology 2nd Edition. Harper Collins, New York. 1995.

2. Cowan, Marie J. (1995). Measurement of Heart Rate Variability. Western Journal of Nursing Research, 17(1), 32-48.

3. Drake, Geoff. (1992). The Lactate Shuttle. Bicycling, 33, 36-37.

4. Sahlin, K, R.C. Harris, B. Nyland and E. Hultman. (1976). Lactate content and Ph in muscle samples obtained after dynamic exercise. Pfluers Arch., 367, 143-149.

5. Gollnick, P. and L. Hermansen. (1974) Biochemical adaptations to exercise: anaerobic metabolism. Exercise Sports Science, 1, 1-43.

6. Tesch, P.A., and J.E. Wright. (1983) Recovery from short term intense exercise: Its relation to capillary supply and blood lactate concentration. European Journal of Applied Physiology, 52, 98-103.

7. Brooks, G.A. and G.A. Gaesser. (1980) End points of lactate and glucose metabolism after exhaustive exercise. J. Appl. Physiol: Respirat Environ Exer Physiol. 49(6), 1057-1069.

8. Urhausen, A., B. Coen, B. Weiler and W. Kindermann. (1993) Individual anaerobic threshold and maximal lactate steady state. International Journal of Sports Medicne. 14(3), 134-139.

9. Richardson, M.T., et al. (1996) Recovery from maximal swimming at the predicted initial onset of blood lactate accumulation. Journal of Swimming Research. 11, 33-35.

10. Darr, Kevin C. et al. (1988) Effects of age and training status on heart rate recovery after peak exercise. American Journal of Physiology. 254 (2P+2), H340-H343.

11. Winer, B. J. (1971) Statistical Principles in Experimental Design, 2nd Edition. McGraw-Hill, New York.

12. Zar, S.H. (1996) Biostatistical Analysis, 3rd Edition. Prentice-Hall, New Jersey.